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DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

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DISEASES of the NERVOUS SYSTEM
(PUBLISHED MONTHLY)

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

The Organic and Physiologic Factors in Mental Defect*

HAROLD D. PALMER, M.D.
Philadelphia, Pennsylvania

PART I

Introduction

Mental defect is limitation or loss of intellectual capacity caused by hereditary fault, by indirect influences or injury or by destruction of brain tissue experienced before adolescence. Intellectual disability resulting from damage occurring after adolescence is not generally known as defect, but as dementia. Although usually prolonged into adult life, mental defect will be discussed in this review as related to the prenatal and childhood periods, since at these times it can be studied most profitably. Etiological factors are more readily ascertainable than in later years and remedial measures are more likely to prove effective if applied at the earliest possible age.

Detailed classification of all types of mental defect is beyond the scope of this contribution. Little additional light would be shed on the subject by a review of known clinical features, or by further elaboration of accepted categories or of diagnostic criteria. It will be more constructive to develop a practical grouping according to etiology, since understanding of causation is the foundation of preventive measures and therapeutic progress. The subject of defect offers a limited number of promising aspects, and emphasis will be laid on those branches of study in which our knowledge of the disorder has been significantly advanced.

In recent years medical science has accomplished the transfer of many pathological entities in various fields of medicine from the hereditary or congenital primary group to the group of biochemical or physiological secondary defects, the latter being more visible as to etiology, more preventable, and in many instances more responsive to treatment. Contemporaneous studies of mental defect show that in this field also the distinction between primary and secondary etiological factors has become less definite. On the one hand, much has been learned concerning the nature of many chemical and structural faults occurring in hereditary disorders, and on the other hand we have acquired greater understanding of the multiplicity of toxic, infectious, endocrine, traumatic, nutritional and other prenatal factors operating on the germ cell and tending to weaken or distort its developmental potentiality. Differentiation between protoplasmic defect of strictly hereditary nature and the influence of accidental factors on embryonic structures is a most complicated task. In postnatal life, the separation of hereditary etiology from the metabolic, physiologic or emotional injuries leading to mental deficiency is equally difficult.

In working out an etiologic classification it has therefore been found impracticable to depend solely on the traditional division into primary and secondary causative factors as

*From the Institute of the Pennsylvania Hospital and the Department of Psychiatry, Woman's Medical College of Pennsylvania, Philadelphia, Pennsylvania.

formulated before advances in biological research had caused the meaning of these differentiating terms to become modified. It will be more accurate, therefore, in view of the fluid state of our present knowledge, to use the heading "Hereditary or as Yet Unknown Causes" in place of the customary term "Primary Causes."

A long step forward in the attack on mental defect has been the study of the pathogenic factors operating within the pregnant mother and within the newborn infant. This study yields data as to causation as well as preventive management. Such fundamental research as that conducted by several embryologists in attempting to influence fetal development by a variety of artificial means has provided a stimulus to research in the whole field of mental defect. As Ford points out, the chilling of the fertilized egg for a brief interval has produced defects in development which are of great interest. It has been shown by this means that local defects of development may be produced by temporary inhibition of embryonic oxygenation. If, during the period an organ is developing most rapidly, the metabolism of the embryo is inhibited by cold, or by being placed in some unfavorable medium, the organ will never have an opportunity to attain its proper growth and will be absent or rudimentary. By careful timing of interference, based on knowledge of the chronology of development of the different embryonic structures, Stockard has been able to produce defects of almost any organ at will. It is possible, therefore, that spontaneous defects of development may be the result of transient disturbances of embryonic metabolism possibly caused by defective implantation or infarction of the placenta. Maternal toxemia must also be considered. The theory is especially appealing in cases in which there is no evidence of morbid heredity and particularly in those in which the anatomical findings seem to indicate that the development of the cerebral cortex has stopped abruptly at a certain stage of fetal life. Also important is the increasing use of the electroencephalograph, which makes possible differential diagnosis (Lindsley and Cutts; Bradley; Palmer and Hughes) between defectives with cortical dysrhythmia (traumatic, epileptic or inflammatory) and those whose cortical rhythms are normal, as has been found by Keezer to be the case with mongolian and "hereditary" defectives. This differentiation has pointed the way to better adaptation of treatment.

Mental defects caused by inherent faults in the protoplasm resulting in structural anomalies of the central nervous system are for the most part unapproachable therapeutically. Those of toxic, traumatic and infectious origin also have somewhat limited corrective possibilities, but as progress is made in our understanding of the origins of intrauterine damage it may be expected that preventive care in the prenatal stage will become more effective. During the past few years the most satisfactory advance in the clinical study of mental defect has been in the disorders caused by endocrine imbalance. In certain types of cases of this kind, treatment by administration of glandular substances offers appreciable promise of amelioration.

Etiological Classification of Mental Defect

I. *Mental Defect Due to Infections of Infancy and Childhood*

- A. Congenital Syphilis
- B. Juvenile Paresis
- C. Encephalitis
- D. Meningitis
- E. Central Nervous System Complications of Other Illnesses.
  1. Mumps
  2. Pneumonia
  3. Measles
  4. Influenza
  5. Pertussis
  6. Scarlet Fever
  7. Poliomyelitis
  8. Chorea
  9. Other Diseases of Infancy and Childhood

II. *Mental Defect Due to Cerebral Trauma*

- A. Intrauterine or Fetal Trauma
  1. Congenital Athetosis
  2. Spastic Diplegia and Paraplegia
  3. Porencephalic Cyst
  4. Epilepsy
  5. Anoxic Encephalopathy
  6. X-ray Damage to Fetal Neural Structures
- B. Birth Trauma
  1. Hemorrhage
     a. Cerebral

b. Extradural
c. Subdural
d. Subarachnoid
2. Asphyxia with Infantile Anoxic Encephalopathy

C. Trauma in Infancy and Childhood
1. Cerebral Concussion
2. Cerebral Asphyxia

III. *Mental Defect Due to Toxic States*

A. Intrauterine or Fetal Toxemia
1. Maternal Toxemia of Pregnancy
2. Maternal Chemical Toxemia
a. Malnutrition and Vitamin Deficiency
b. Placental Transmission of Drugs
c. Anoxemia

B. Toxemia in Infancy and Childhood
1. Icterus Gravis Neonatorum
2. Malnutrition and Vitamin Deficiency
3. Accidental Poisonings (lead, carbonmonoxide, etc.)

IV. *Mental Defect Due to Developmental Structural Pathology in the Central Nervous System*

A. Porencephaly
B. Anencephaly and Other Forms of Lack of Cerebral Structure
C. Microcephaly
D. Macrocephaly
E. Hydrocephaly
F. Craniostenosis
G. Cranium Bifidum
H. Neurofibromatosis
I. Tuberous Sclerosis
J. Cerebral Dysplasia of Prematurity
K. Other Forms

V. *Mental Defect Due to Endocrine Disorders*

A. Hypothyroidism
1. Central Nervous System Damage of the Fetus Due to Maternal Hypothyroidism
2. Congenital Hypothyroidism or Cretinism
3. Infantile Hypothyroidism
4. Juvenile Hypothyroidism

B. Pituitary Disorders
1. Frolich's Syndrome
2. Lawrence, Moon, Biedl Syndrome

C. Adrenal Cortical Hyperplasia (pubertus praecox or virilism)
D. Combined Types

VI. *Mental Defect Due to Hereditary or as Yet Unknown Influences*

A. Amaurotic Family Idiocy
1. Infantile or Tay-Sachs
2. Late Infantile or Janksy-Bielschowsky
3. Juvenile or Spielmeyer-Vogts
4. Adult of Kufs

B. Phenylpyruvic Oligophrenia
C. Neuromuscular Disorders
D. Friedreich's Ataxia
E. Schilder's Disease (Schilder-Fox)
F. Huntington's Chorea
G. Wilson's Disease (Hepato-lenticular)
H. Idiopathic Epilepsy
I. Mongolism
J. Other Types

VII. *Mental Defect Due to Indirect or Environmental Causes*

A. Psychological Disorganization
1. Infections or Trauma Not Directly Involving the Cerebral Structures
2. Physical Handicaps
a. Impaired Vision
b. Impaired Hearing
c. Malnutrition
d. Anemia
e. Motor Disabilities
3. Emotional Factors
4. Deprivation

Based on classification into the above seven groups a rearrangement of the etiological data contained in Barr and Whitney's statistical study of 5000 mental defectives (1930) shows the following: (1) Mental defect due to infections of infancy and childhood, 16.32 per cent; (2) Mental defect due to cerebral trauma, 11.20 per cent; (3) Mental defect due to toxic states, 4.42 per cent; (4) and (5) Mental defect due to developmental structural pathology in the central nervous system and to endocrine disorders, 28.46 per cent; (6) Mental defect due to hereditary (or as yet unknown) influences, 38.60 per cent; (7) Mental defect due to indirect or environmental causes (deprivation, abuse, neglect and exposure), 1 per cent.

I. *Mental Defect Due to Infections of Infancy and Childhood*

A. *Congenital Syphilis*—Studies of the problem of congenital lues in relation to mental

defect have been undertaken in the majority of instances from the viewpoint of incidence of congenital syphilis among institutionalized mental defectives. Conclusions drawn from this approach are often quoted erroneously to indicate the importance of syphilis in the etiology of mental deficiency. Obviously there can be no assurance that the coexistence of syphilis and mental defect is one of cause and effect. Moreover the figures obtained from institutional material are in such wide disagreement that they cannot be used as the basis for any accurate statistical statement. Benda and Tadgell found that 6 to 7 per cent of 2000 mentally defective children had congenital lues, while other authorities hold that 25 per cent or more of all mental defectives show positive Wassermann reactions. Estimates of the incidence of mental defect among congenital luetics are even more at variance. One authority recently stated that less than 4 per cent of children with congenital lues had any intellectual defect, while others hold the incidence to be as high as 40 or 50 per cent. No more conclusive statement can be made than that from 4 to 40 per cent of congenital luetics are mentally defective, and that this syphilitic group accounts for from 6 to 25 per cent of the total number of defectives. These figures are not only unsatisfactory as statistics, but also fail to indicate the destructive scope of congenital syphilis in the succeeding generation. Benda and Tadgell, in a study of 100 luetic families, found that of 337 born alive 104 died in infancy, giving a mortality rate of 30 per cent, or 6 times the normal rate. It has been found that in congenital luetics without central nervous system involvement there may be no intellectual defect, and in many early cases where central nervous system involvement has occurred there may be only the general signs of failure of neurological integration and of intellectual growth evidenced by retardation in learning to crawl, walk, etc. Probably not more than half of the cases of congenital syphilis develop neurological or mental defects, so it must be concluded that the presence of syphilis in mental defectives is not a complete explanation of the cause of intellectual retardation and cannot be taken as a diagnosis of exclusion. In cases showing mental defect, the presence must be postulated on a deciding factor which may or may not be related to the syphilitic infection.

As our diagnostic knowledge of syphilis has progressed, the long established tendency to attribute to lues nearly all congenital mental defects and most of the neurological disorders found among mental defectives, has given place to a more scientific point of view, which demands the evidence of a positive Wassermann reaction on more than one occasion, and does not depend on the presence of the so-called luetic stigmata. These traditional diagnostic signs of congenital lues, the Hutchinson teeth, karatitis, saddle nose, bossing of the frontal bones and scarring of the skin and mucous membranes at the corners of the mouth, are in fact rare findings and useful as diagnostic aids rather than criteria. Acceptance of a false positive serological test is to be guarded against since positive reactions tend to occur occasionally in respiratory infections, skin diseases, pertussis, scurvey, etc. It is advisable to use both the Wassermann and Kahn tests in diagnosing congenital lues. Clifton and Heinz report a fairly high percentage of false positive Kahn tests but no false Wassermann tests in a group of children with lues.

The mental defect associated with or caused by congenital lues need not be regarded as completely irremediable, although Levin and his associates believe that the congenital form of this disorder produces widespread changes which are more resistant to treatment than acquired syphilis. The aim, of course, should be the early detection and treatment of the maternal infection before delivery. In the new-born infant diagnosis is advisable at the earliest possible moment by every means available: study of the placenta, cord, bone x-ray and serological reactions of mother and child. Treatment of the pregnant mother is indicated by means of neoarsphenamine or the more recently developed mapharsen by intravenous injections, not only in the hope of improving the physical state but also in an attempt to minimize the destructive or retarding effects upon the mental capacity of the fetus. Levin and his associates treated congenital and acquired syphilis in children by means of massive doses of mapharsen given by continuous intravenous drip over a period of 5 days. The children so treated had no untoward toxic reactions and there was no mortality. All those with acquired syphilis

and 37.5 per cent of the congenital luetics showed serological reversals.

B. *Juvenile Paresis*—The early diagnosis of juvenile paresis is often very difficult inasmuch as the condition may exist without evidencing any recognizable symptoms until the teen ages. Menninger has found that 60 per cent of children develop normally up to the time of onset of the disease and that only 40 per cent show real mental retardation. It is not uncommon for the child to go as far as junior high school with an excellent record only to break down suddenly with what looks like a schizophrenic or severe psychoneurotic reaction. The most commonly seen clinical symptoms are confusion, inadequate emotional response, restlessness, visual and auditory hallucinations, delusions, memory loss and periods of excitement. Occasionally the disease is first suspected because of convulsive seizures resembling those occurring in adult paresis.

Case example of mental deficiency due to juvenile paresis—A girl of fifteen was brought to the Institute for study. The history in brief was that she was nervous, was constantly picking at her scalp and pinching her skin, that she had become withdrawn and seclusive, and did not mix with other children. Her history prior to age 13 was that she had been better than average in school work and had been a wholesome outdoor girl, interested in other children, and considered by her teachers to be a very promising child. The parents attributed her difficulties to the onset of menses at age 14, at which time she gained weight rapidly and changed in appearance. She gradually became careless about the house, behaved in a preoccupied manner, collected bits of paper, and wadded clothing up into small compressed bundles and tucked them away behind furniture, etc. The blood Wassermann reaction was 4 plus, and the spinal fluid study showed a positive Wassermann reaction and a paretic colloidal gold curve.

The course of untreated cases is that of very rapid deterioration with ultimate dementia. The average course from the first appearance of the disease to its terminal state is slightly under five years. Neurological disintegration is likewise rapid with the appearance of convulsions, speech defects, hemiplegic states, contractures and terminal emaciation. Treatment of juvenile paresis by means of malarial therapy or hyperthermia induced by diathermy or by other means, together with tryparsamide or other arsenicals, has brought about only a slightly improved prognosis. Cure, with restoration of intellectual capacity, is not to be hoped for, but the rapid physical and mental dilapidation may be prevented in a fairly satisfactory number of cases. Menninger in a survey of 653 cases found 144 treated patients. Fourteen per cent were markedly improved, 25 per cent were slightly improved and 61 per cent unimproved. The method of treatment in the majority of cases was by malarial therapy and some combination of chemotherapy, either tryparsamide or arsphenamine. The results when compared with success in adult paresis are discouraging. The prognosis is related to the early recognition of the disorder and those patients in whom the clinical symptoms appeared after a period of normal development responded better to therapy than those who were initially defective. The most effective method of treatment is by induced malaria, with tryparsamide and bismuth, or a series of arsphenamine injections. Intensive treatment over a long period of time is recommended.

C. *Encephalitis*—In the past twenty years the number of mental defectives contributed by encephalitis has been reasonably well estimated and the now well known characteristic behavior problems following encephalitis flood the mental hygiene clinics, social agencies and child guidance clinics. A number of unanswered questions exist in relation to the problem of the intellectual defects resulting from the disease since only some of its victims suffer from true intellectual impairment. The more frequent damage seems to be limited to subcortical structures and to the individual's capacity to inhibit his anti-social impulses. Penrose has stated that the intellectual defect caused by encephalitis is likely to be on a somewhat higher level than the average institutional type. More important than epidemic encephalitis in infancy or childhood in the etiology of mental defect is the damage done to the neural structures when the virus disease occurs in the mother during pregnancy. Demonstrations of encephalomyelitis in the fetus following measles, scarlet fever and other maternal infectious states, even when the mother appears to have escaped

central nervous system complications, have been reported by various investigators. There is no clear-cut answer to the question why the fetus sometimes escapes injury even in the presence of severe maternal encephalitis. However, when in such cases there is no demonstrable fetal encephalitis there is unquestionably some unfavorable influence on the nervous system of the child. Some authorities feel that the ill effects of maternal encephalitis on the fetus are not usually more severe than in the average febrile disease. Bassoe found no characteristic lesions in the central nervous system of a seven months' fetus dying in utero as a result of the mother's death from epidemic encephalitis. Jorge, on the other hand, reported a case of transmission of the disease from the mother to the fetus. There are numerous cases in the literature and in the experience of neurologists in which encephalitis occurring prenatally or in early life is known to produce such grave damage that severe intellectual impairments result. In all these children suffering from intellectual retardation there are some of the characteristic post-encephalitic neurological signs even though the typical Parkinson's syndrome may be absent. In cases where obvious neurological disorders are absent some minor evidence of organic disease can be detected on careful examination. The progressive nature of the organic disorder and intellectual defects are distressing features of the condition. In the past five years the electroencephalographic studies of mental defectives have advanced our knowledge of the unrecognized or sub-clinical encephalitic processes in children. In our recent study of mentally retarded children at the Institute of the Pennsylvania Hospital a number of cases attributable to early encephalitis were found.

Case example of sub-clinical post-encephalitis with mental defect and behavior problem —A girl of 10 years was brought to the Institute because of severe emotional maladjustment in the home and at school and failure to progress scholastically. Pregnancy before her birth had been normal and delivery was spontaneous after a normal 8-hour labor. At 11 months of age an illness had occurred characterized by complete anorexia, restless delirium and later stupor from which she could be aroused only with difficulty. After 3 weeks of illness she improved and appeared well at the end of 5 weeks. She was retarded in learning to walk and did not begin to talk until age 5. At 3 years she had severe whooping cough complicated by convulsions. The parents noted many odd reactions from age 2 years. She never cried when hurt, was extremely irritable and had breath holding spells to the point of severe cyanosis when she was angry and some incoordination of the right hand. Imbalance of ocular muscles was noted at age 3. At 4½ years pneumonia occurred with fevers as high as 106°. During the ensuing years behavior became more and more restless and powers of concentration and attentiveness appeared to be lacking. Conduct was erratic, there were rather troublesome demonstrativeness with strangers and occasional apparently unmotivated spells of crying. Menses began at 9½ years and fainting spells accompanied each period.

The electroencephalogram was interpreted by Dr. Joseph Hughes as follows: There are large abnormal waves seen in all cortical leads. These vary from 3 to 7 per second in rate and from 50 to 100 microvolts in amplitude. They indicate a generalized cortical involvement and are similar to the findings seen after encephalitis.

The psychometric tests gave the results listed below:

Whether or not there is demonstrable intellectual damage or focal neurological signs, the total clinical picture includes the classical behavior pattern of post-encephalitis. This

Stanford-Binet	Mental Age	6 years, 8 months
	I. Q.	64
Kohs Block Design Test	Mental Age	6 years
	I. Q.	59
Witmer Formboard Witmer Cylinder Paterson and Dearborn Formboards	Mental Age	6 years
	I. Q.	59

is marked by restlessness, impulsive advances, episodes of wanton destruction, display of undue affection, assaultiveness, etc. The reactions of the lower level are intensified and emotional responses and responses to environmental conditions are to a large extent uninhibited (Bond). In general, those children who during the acute encephalitic processes have the evidence of most widespread organic involvement also present the most severe post-encephalitic behavior and the most advanced intellectual defect. In certain instances in the literature it has been stated by various authorities that the degree of behavior disorder was such that the child had to be deprived of normal educational opportunities and this may have accounted for some portion of the intellectual failure.

The use of benzedrine sulphate in the treatment of post-encephalitic disorders has been a real advance since it appears to do more than bring about symptomatic relief. It appears to alter the cerebral chemical state in a way which favorably effects the electrical potentials with the result that the electroencephalogram tends to return to normal.

D. *Meningitis* — The acute symptoms of meningitis need not be reviewed here since the classical syndrome is well known to the medical world. Parents and the physician will do well to be on their guard after the acute symptoms have subsided and during the convalescent stage of the illness, since it is not rare for an intellectual defect to go undiscovered until a year or longer after the organic damage has been done to the nervous system. The delay in return to normal intellectual function may be attributed to a "slow convalescence" and the parents or the doctor may resassure themselves that the child will be "all right after he has been up and about in the fresh air and sunshine." In general, the cases in which intellectual defect is most likely to occur are those having numerous symptoms of severe organic nervous system involvement during the acute course of the disease. Convulsive seizures of local or generalized type, paresis or spasticity of the extremities, strabismus, etc., are some of the clues to severe organic damage. Often a mental defect has been marked by a change of personality in the child and it is advisable for the practitioner to be alert in detecting and measuring the quality as well as the quantity of intellectual impairment if the child fails to respond after a normal convalescent period. In some instances there may be no actual intellectual loss but merely a failure to develop further in mental age after a devastating illness.

E. *Central nervous system complications of other illnesses*—Medical practitioners have recently recognized a form of meningoencephalitis occurring in mumps and in some reports the incidence is said to be as high as 4 per cent. The acute symptoms are those of any encephalitic process, namely, nausea, vomiting, severe headache, diplopia, abnormalities of the reflexes and occasionally a positive Kernig sign. The spinal fluid shows an increase in the number of cells ranging from 100 to 1000. Recovery is the rule within a few weeks. Tabor and Newman report a follow-up study of a number of patients. The majority had persistent irritability, nervousness, enuresis, attacks of fainting and a number failed to do satisfactory work after their return to school. The possibility of permanent involvement with damage to the intellectual functions is suggested by their study. Pneumonia, influenza, scarlet fever and measles may also have a degree of central nervous system involvement, either of the meningitis, encephalitis or combined types. Measles encephalitis was found by Peterman and Fox in a study of 27 cases of this disorder to occur usually on the fourth day after the rash. It was characterized by coma, convulsions, rigidity of the neck, spasticity of the extremities and by dilated pupils. The mortality rate was 33⅓ per cent. Only one patient became hopelessly mentally defective in the immediate post infectious period. Later sequellae were not reported.

Cerebral damage resulting from pertussis has recently been reported by Lurie and Levy. They made special studies of a group of 58 children who had suffered from pertussis at the age of 2 years or younger. Twenty-five of the cases had definite clinical neurological findings of pyramidal and extrapyramidal nature. Only 6 cases had electroencephalographic tracings and 5 of these were definitely abnormal, showing dysrhythmias characteristic of epilepsy, cortical atrophy or other abnormal states. Twenty-four cases showed less than average intelligence, a much higher incidence than in the average of the cases

studied in Lurie's clinic. The figures were as follows: 9 were subnormal, 5 were borderline feebleminded, and 10 were definitely mentally defective. All of the children showed abnormalities of behavior; 8 were classified as prepsychotic or psychotic, 6 showed progressive organic deterioration, while 17 exhibited typical post-encephalitic behavior disorders. The disorders of intellectual function and behavior appeared to result from the various types of organic destruction grouped under the term whooping cough encephalopathy. Complicating encephalomyelitis has been reported in cases of chicken pox, poliomyelitis, Sydenham's chorea, erysipelas and scarlet fever. The consequences of this inflammatory or toxic nervous system disorder depend on its severity and duration, the degree and duration of the stupor and the severity and frequency of convulsive seizures indicating the extent of the permanent nervous system damage.

II. *Mental Defect Due to Cerebral Trauma*

A. *Intrauterine or fetal trauma*—There is growing evidence that many of the so-called congenital mental defects hitherto believed to be hereditary arise from prenatal trauma and that injury after conception may result from numerous complications of pregnancy.

The role of intrauterine infections in relation to cerebral birth conditions has been reviewed extensively by Alpers and Patten and they leave little doubt as to the transmission to the infant in utero of a number of infectious processes resulting in extensive damage to the brain structures. They believe that cerebral diplegias, congenital athetosis, and extrapyramidal lesions which are commonly attributed to birth trauma and are known to be associated with mental deficiency, may have their origins in some infectious or traumatic damage in utero long before birth. Definitely demonstrable damage occurs in typhoid, paratyphoid, pneumonia, anthrax, scarlet fever, measles, poliomyelitis and malaria. It has been shown that the streptococcic infections damage the placenta or are transmitted through the placenta to the fetus. Prenatal trauma, as well as infectious and toxic states, favors the occurrence of porencephalic cysts. Alpers has discussed the various factors operating during pregnancy which predispose the fetal tissues to damage at birth. In addition to infectious disease, syphilis, prematurity, eclamptic states and drug toxemias are among the agencies which seriously affect the vulnerability of the fetus and indicate that cerebral birth conditions traditionally attributed to birth trauma may in fact be only partially the results of physical injury at birth. This supposition is supported by evidence that prenatal organic brain involvement among mentally defective patients is far more common than ordinarily thought. Patten and Matthews, for example, examined 350 cases of mental defect and report that 50 per cent of all cases with I. Q.'s below 50 had definite neurological symptoms and signs. Of these, 36.2 per cent had epilepsy. They found that there is a relation between neurological involvement and the depth of the intellectual defect and that in only 4.2 per cent of the cases were the neurological conditions postnatal in origin. This finding is supported by a study made by Brander of 72 cases of cerebral trauma observed in Caesarian deliveries. He describes tentorial tears, hemiplegias, diplegias, epilepsy, mental deficiency and other conditions generally attributed to birth injury, which were seen in cases where the possibility of birth injury was absent. Sachs found that the most common prenatal causes of hemiplegia with mental defect were porencephalic cysts, defective development of the pyramidal tracts and cerebral agenesis. Acute trauma to the mother during pregnancy was found to be a possible cause of spastic paralysis, convulsions and mental deficiency. It is believed that the majority of cases of cerebral diplegia result from some maldevelopment in utero.

Fetal anoxemia of traumatic origin is also a possible cause of mental defect. It may be produced by maternal uterine bleeding resulting from dislocation of the placenta insufficient to cause miscarriage but sufficient to cause oxygen deprivation in the fetal cellular tissue.

Damage by x-ray treatment to the fetal neural structures has been thought to be an important factor in the production of certain types of arrest of neural growth. Doll and Murphy in a report of 25 defective children irradiated in utero appear to show definitely that in the early months of pregnancy x-ray damage to the embryonic neural structures can result in failure of brain development and

in the production of microcephaly.

It is a fair conclusion from these findings that many organic disorders of the central nervous system are due to a failure of, or accident to, neural evolution in intrauterine life, but the many possible causes of this pathology are still a matter for further research and analysis.

The whole group of cases with hypertonic disorders, rigidities, paraplegias, diplegias, athetoses, spasticities, etc., is offered some help by the painstaking re-education efforts designed by Phelps and others. The basic plan is to assist the patient to gain power of control over the extremities by voluntary relaxation and activation (innervation) of single muscle groups. Many children are able in this way to acquire an encouraging degree of control and coordination. Putnam has performed the operation of anterior and anterolateral cordotomy in a large series of cases with success. Bucy and Buchanan have devised the surgical procedure of extirpation of a cortical area of the brain (Brodmann's area 6) and relieved athetosis although the affected limb remained paralyzed.

B. *Birth trauma* resulting in either hemorrhage or asphyxia is one of the most frequent and obvious causes of mental defect. Unless accompanied by hemorrhage, the compression and distortion of the cranium occurring at birth cause no permanent damage. Estimates of the incidence of cerebral birth injuries vary considerably, but in a large statistical accounting between 1 and 2 per cent of all babies died of birth injury. Of all autopsies in infants dying from birth to 5 weeks of age, 65 per cent were found to have cerebral vascular lesions of supposed traumatic origin. Forty per cent of those stillborn or dying the first three days had some intracranial traumatic condition. Roberts reports an incidence of 0.7 per cent of intracranial hemorrhage occurring in nearly 2000 deliveries. The presence of subarachnoid hemorrhage in the new born likewise tends to indicate that extensive trauma may be unrecognized in that there may be no localizing neurological syndromes such as hemiplegia, diplegia, etc. It is concluded that the gross signs of birth trauma such as distortions of the cranium, monoplegia, paraplegia and spasticity, are not always the only methods of determining the presence or absence of intracranial damage.

The most frequent immediate causes of cerebral hemorrhage at birth were found by Sachs to be prematurity and difficult labor.

The prognosis in cases of cerebral or intraventricular hemorrhage in the new born is extremely poor. The vast majority of such cases die within a few days. The incidence of mental defect among the survivors has not been determined statistically, but is known to depend to a large extent on the size and location of the hemorrhagic lesion. It is likely that in the infants who survive, the hemorrhages are petechial in character. The mental deficiency resulting from such widespread multiple foci is usually complicated by convulsive seizures, hemiplegia, diplegia, etc.

Extradural hemorrhage at birth is usually caused by fracture of the infant's skull. The occurrence is rare and if the bleeding is not stopped by ligation of the vessel, it is invariably fatal. The symptoms are the same as those in adult extradural bleeding, except that the fontanelles bulge as a result of increasing intracranial pressure. If, after ligation of the bleeding vessel, the clot is not evacuated, severe compression damage to the brain with resulting hemiplegia, mental defect or convulsions will occur.

Subdural hemorrhage is usually caused by birth trauma, but scurvy and syphilis are contributory factors. Alpers states that the most common injury is a tearing of the tentorium, or laceration of a dural vein. The blood clot may be very extensive and may be bilateral. If the clot is not removed, it undergoes slow organization and may be enclosed by thickened fibrous membranes or may become calcified, giving rise to irritation and destruction by pressure upon the underlying cerebral cortex. Complete hemiatrophy was caused by extensive hematoma in cases reported by Ford. Convulsions, various paralytic symptoms and mental defects of varying degrees usually result. Diagnosis and localization can be accurately established by air encephalogram or by the insertion of a needle through the dura and aspiration of blood.

Subarachnoid hemorrhage is believed by Alpers to be of fairly common occurrence in new born infants. Estimates by various investigators as to its incidence vary from 0.05 per cent to as high as 11 per cent. Fay states that 8 to 13 per cent of normally delivered

babies suffer subarachnoid hemorrhage, but that only 2 per cent of these develop neurological signs. Its role in the production of mental defect is debatable. It is believed by some that subarachnoid bleeding produces a clogging of the spinal fluid escape pathways giving rise to a damming up of cerebrospinal fluid, increased intracranial pressure, cortical irritation with generalized epileptiform convulsions and atrophy of the cortical cells resulting in mental defect. Others are of the opinion that blood in the spinal fluid is relatively innocuous, is absorbed and produces no pathological consequences.

The role of asphyxia at birth in the production of encephalopathy has assumed increasing importance. Its incidence has not been determined, but Cole, Kimball and Daniels found the incidence greater in premature and first born infants, in the offspring of mothers suffering from severe illness, in cases of trauma sustained during delivery and in those cases in which sedatives and anesthetics were used in large amounts during labor. The exact relation of mental defect to birth asphyxia is undetermined, but it is known that in cases where asphyxia has existed sufficiently long it will produce permanent damage to the ganglion cells of the cerebral cortex as well as impairment of mental capacity. Alpers states that the duration of such anoxemia necessary to produce irreparable damage varies in the different areas of the brain, the cerebral cortex being the most vulnerable and the cells of the medulla the most resistant. In severe cases of traumatic complications accompanied by asphyxia the damage to brain tissue may be the result of petechial hemorrhage throughout the brain rather than of the anoxemia alone. The most common result of asphyxia, however, is cerebral cellular degeneration, which may be widespread. In a study of 500 children with mental deficiency, convulsions, spasticity and other conditions resulting from central nervous system damage Schreiber found that apnea at birth with varying degrees of asphyxiation appeared to be the causative factors.

The most common causes of asphyxia are premature separation of the placenta, cord complications, toxemias of pregnancy, analgesic drugs and delayed breathing of the new-born infant.

It is unsatisfactory to discuss cerebral trauma in utero and cerebral trauma at birth exclusively as two separate entities, since in some forms criteria for distinguishing between them are lacking, and in others the two pathologies are known to be inextricably interwoven. With no special reference to mental defect, Alpers has made a useful classification of the etiological factors in cerebral traumatic birth conditions based on this differentiation which indicates the close relationship between the two entities.

The recognition of mental defect due to cerebral trauma before or during birth is usually delayed until late infancy, when it is as a rule obviously associated with neurological symptoms such as hemiplegia, monoplegia, paraplegia, epilepsy and athetosis. In a study of 33 children four to fifteen years of age with definitely established prenatal and birth injuries, Ford found these forms of impairment as well as mental defect. In the presence of one or more of these conditions mental defect is to be suspected and it may occasionally occur without other symptoms.

The use of the electroencephalogram in determining the presence of organic damage due to birth trauma, asphyxia or infection is valuable since the electrical tracings of a number of such cases recently studied by the author showed the presence of abnormal electrical rhythms, although no evidence in the history or in the neurological study indicated the presence of an organic factor in the mental defect. The electrical potentials have been studied in certain types of mental defect by Keezer, who, in 1936, made electrical tracings in 41 mongols, 18 hereditary mental defectives and 22 normals. He found that these types of defectives do not differ from the mentally normal in their electrical tracings. However, where abnormal rhythms are demonstrated, it is our belief that one is justified in concluding that the underlying cortical damage is either casutive or materially contributory to the intellectual deficiencies.

C. *Trauma in infancy and childhood* — Nearly all parents bringing problem children to a physician date the onset of the mental retardation or misconduct from a blow on the head. Obviously this has been much overemphasized in the past and still is in the minds of the laity. Nevertheless, there is a well-recognized pattern in the defects asso-

ciated with trauma to the head in childhood.

Strauss and Savitsky believe that there is too much tendency to disregard the traumatic origin of behavior disorders and mental defect if there is no significant paralysis or neurologic syndrome in evidence. They believe that unconsciousness, diplopia, restlessness, and delirium are some of the evidences of widespread cerebral damage, and that careful neurological and psychiatric examinations immediately after cerebral concussion show that there are evidences of organic nervous system derangement even though it is only transient.

Blau studied 22 children with verified head injuries and found that their predominant behavior characteristic was unrestrained antisocial conduct with little alteration of total intellectual capacity. The reaction probably can best be summed up as "loss of inhibitory control." Twelve of the 22 showed delinquent behavior of chronic recurring nature which had not been present before the injury and which appeared in each case within one year after the trauma. The diagnosis of posttraumatic encephalopathy rests upon history of trauma with or without skull fracture, evidence of unconsciousness, or other signs of concussion; headache, which is aggravated on bending or stooping; attacks of vertigo and unsteadiness; memory defect due to poor retention; inability to adjust to new situations; fatigability and in general a restless uninhibited behavior pattern.

Strecker defines the traumatic constitution in childhood in approximately these terms, but he adds sexual aggression and calls attention to the marked resemblance of the traumatic behavior disorders and defect to postencephalitic disorders. Traumatic defect conditions, as described by Strecker, occur with the more profound cases of head trauma and are usually revealed by amnesia, episodes of confusion, epileptoid states, severe disorders of attention, and inability to progress scholastically. Clinically the differential diagnosis between traumatic behavior disorders and the functional (hysterical) types rests chiefly upon the evidences, however slight, of cerebral damage and is evidenced by the fact that the child prefers to keep away from all forms of noise, excitement and amusement in marked contrast to the hysterical, postencephalitic or, in fact, the normal child. The electroencephalogram now makes differential diagnosis an exact matter. The presence of typical cortical dysrythmia is easily established months or even years after the original concussion. Experience cautions us to think, not altogether of demonstrable organic changes in the brain, but in terms of total reactions of the individual child. The personality and intellectual deterioration associated with severe traumatic encephalopathy must be considered irreversible.

Asphyxiating disease in the first years of life has been studied by Bradley, who correlates its incidence with intellectual and behavior deviations and with electroencephalographic changes. The far-reaching organic damage of anoxemia occurring in early infancy is stressed by Chornyak, especially with reference to pertussis and other acute infectious diseases of childhood. There is partially substantiated evidence that in children resuscitated from drowning or suffocation cerebral damage may be caused by asphyxia.

Strauss and Werner suggest that at least in part some cases of intellectual impairment seemingly due to organic injury at birth or in early infancy may be the result of lack of adequate educational effort. Children with a definite lack of sensori-motor organization will lag behind the other children in any work which demands the coordination of motor and sensory functions. They caution against allowing such children to deteriorate when their experience with the training of mentally crippled children has demonstrated a considerable degree of success through the use of special methods and special material. They found in an examination of an unselected group of moron and borderline children that 20 to 25 per cent suffered from brain lesions notwithstanding the fact that only 3 to 5 per cent showed obvious evidence of gross neurological disorders. In their complete analysis of this group they found a personality fundamentally different from the personalities of mentally defective children in whom no evidence of organic nervous system disease could be demonstrated.

Many causes of mental defect mentioned in this section, such as intrauterine infections, toxic states, epilepsy and maldevelopment, are discussed at greater length under other headings.

III. *Mental Defect Due to Toxic States*

A. *Intrauterine or fetal toxemia*—Maternal toxemia caused by eclamptic states, nutritional deficiencies, drug administration, etc., may later be shown to be a significant factor in the production of some types of mental deficiency. Eclampsia not only causes toxic and degenerative changes in the infant brain and increases the danger of fetal anoxemia but also predisposes to prematurity which in itself adds to the hazard of birth injury to the child. The relation between nutritional deficiency and cranial softening in the new-born was studied by Reiss and Bader, who found areas of cranial softening in 32 per cent of 791 new-born infants. It occurred twice as frequently in the Negro race and was more often encountered in the offspring of young mothers 15 to 19 years of age. Diets deficient in calcium and phosphorous and low in vitamin D appeared to predispose to the condition. It is obvious that there is greater likelihood of central nervous system injury during birth in cases of infants with softening of the cranial bones. The prothrombin index of mothers and new-born infants was studied by MacPherson et al. who urged that vitamin K be given to the mother shortly before delivery and to the infant shortly after birth, in all instances of toxemia of pregnancy, premature labor, difficult delivery or in the presence of hemorrhagic disease, severe icterus or anemia, or cerebral symptoms in the mother or infant. The likelihood of traumatic intracranial bleeding in the infant may be materially diminished by such therapy.

Placental transmission of drugs is a problem which must be given more serious study in any evaluation of etiological factors of mental defect. Bromide rash is known to occur in the fetus due to excessive intake of bromides by the mother. The damage done by bromides to the cortical cells of the adult brain is in most instances recoverable, but to what extent high concentrations of bromide can produce permanent damage to the fetal neural structures is an unanswered question. It would seem best to regard bromides and some other cumulative sedative and hypnotic drugs as potentially dangerous to the fetal nervous system, directly through toxic effect as well as indirectly by the production of anoxemic states. The transmission of sulfanilamide and related drugs from the mother to the fetus has been studied by Speert. He has found a markedly injurious effect on the offspring of experimental animals treated with sulfanilamide and demonstrated that intrauterine and post-natal mortality is much greater in the treated cases. He cautions against the prolonged and indiscriminate use of this drug during pregnancy because of the danger of fetal damage. Acute yellow atrophy of the liver and hemolytic anemia have been observed by Heckel and by Ginzler and Chesner in fetuses suffering a high concentration of sulfanilamide from the maternal circulation. In such cases damage to the fetus may also occur through the reduced oxygen carrying power of the blood and the consequent anoxemia. The use of analgesics to diminish the consciousness and memory of labor can be carried to the point of threat to the fetus. A marked delay in respiration was found by Katz and Kaufman in those new-born infants whose mothers had received certain analgesics. Paraldehyde was thought to be the most damaging in that it delayed spontaneous new-born respiration sufficiently to give rise to a considerable degree of anoxemia. In a study of microcephaly Greenfield and Wolfsohn conclude that pathogenic factors acting on the tissue in the earliest stage of development produce a fixation of embryonic evolution, and cite the influence of maternal toxins, alcohol, lues, nutritional deficiencies, etc., as possible etiological factors. Severe or prolonged hypoglycemic states and carbon monoxide poisoning in the mother may also cause fetal cerebral damage. Heavy doses of quinine taken to induce abortion have been known to produce permanent nerve deafness in the infant.

Fetal asphyxia is believed by Schreiber to be a definite cause of a large number of cases of mental deficiency. In a recent study he describes four types of conditions which would lead to inadequate blood supply to the brain of the fetus: (1) the *anoxic* type due possibly to prolonged anesthesia of the mother, strangulation or anything which might reduce the oxygen supply in the fetal circulation. (2) The *anemic* type in which the mother suffered a severe depletion of red cells throughout the pregnancy. (3) The *stagnant* type in which the fetal heart or blood vessels in some manner failed to function adequately

and reduced the supply of oxygen to the fetus. (4) The *histotoxic* type in which some damage to the brain cells is postulated producing interference with the ability to utilize oxygen. In common with other investigators he believes that the use of drugs such as morphine or barbiturates may be a causative factor in this category. The same conclusion was reached by Aldridge and Meredith who, in a study of 271 fetal deaths, expressed the opinion that too much analgesia and anesthesia had been used in cases of cerebral damage or death caused by asphyxia.

B. *Toxemia in infancy and childhood*—The earliest form of toxemia which threatens the mental development of the new-born is that associated with icterus gravis neonatorum, although the presence or absence of resulting cerebral damage cannot be demonstrated until the sequellae have become apparent. In a study made by Kingman and Carlson it is reported that among 657 children with neuromuscular dysfunction characterized by chorea, athetosis, ataxia, spasticity and mental deficiency 45 had survived neonatal jaundice. It is thought that these symptoms represent the so-called nuclear jaundice syndrome. The lesions are limited to the striatopallidal portions of the brain, which are heavily stained with bile pigment during the acute stages of the disease. The later findings indicate that previous destruction of cells has occurred, as shown by loss of nerve cells, demyelination and glial proliferation. Such damage is irreversible and there is no demonstrably effective preventive treatment. MacPherson et al. advise administration of vitamin K immediately after birth to infants with severe icterus, but there is no statistical evidence of its beneficial effect. A large proportion of cases die at the onset of the disease.

Malnutrition and vitamin deficiency in infants and young children may, if continued long enough, produce irretrievable pathological changes in the central nervous system, including varying degrees of mental retardation. Poisonings by carbon monoxide gas, illuminating gas, lead and other chemicals are known to produce mental defect. In particular the widespread and seriously damaging effects on the brain of lead poisoning should be emphasized. Kanner believes that lead intoxication in children is more frequent than is stated in most pediatric or psychiatric textbooks. He emphasizes the importance of suspecting the possibility of lead encephalopathy in all cases of severe degree of mental deficiency. Lead intoxication, involving the threat of possible brain damage, may occur in unsuspected ways, such as that cited by Williams et al. who reported 40 cases of severe lead poisoning in children, caused by inhaling the fumes from a bonfire in which old battery cases were burning. Bucy and Buchanan have found all the signs and symptoms of brain tumor associated with lead encephalopathy and have stressed the importance of careful tests for lead intoxication in all children presenting symptoms of intracranial neoplasm.

(To be concluded in February Diseases of the Nervous System)

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Tick Borne Diseases

(With Particular Reference to Tick Paralysis)

KENNETH H. ABBOTT, M.D.*
Rochester, Minnesota

When bacteria, protozoa and viruses began to be recognized as the causes of many diseases, attention was immediately turned to the possible existence of vectors and hosts from and by which diseases could be spread to man and animals. Among many possible vectors, such as the louse, flea and mite, the tick has risen in the past four decades to a place of prominence. In North America at least five tick-borne diseases have received considerable, and in some instances, exhaustive study in late years; these are Rocky Mountain spotted fever, tularemia, Q. fever, Colorado tick fever and tick paralysis. It is recognized that there are other tick borne diseases, not a few of which are being combated by our overseas troops. One which should be mentioned is tick borne typhus fever.

Rocky Mountain spotted fever, a rickettsial disease, tularemia attributable to bacteria, Q. fever and Colorado tick fever which are probably due to virus, have in late years been the subject of extensive reviews by Parker and others and will therefore not be reviewed again. The less known disease, tick paralysis, is a disease so rapidly fatal and yet so easily cured that it should be recognized by every physician. It occurs in North America, Australia, Africa and possibly in Europe. In the United States it exists in the northwestern states; in Canada it occurs particularly in southern British Columbia, Alberta and Saskatchewan; in Australia it is found in two narrow eastern and western coastal strips. It also is found in South Africa (Cape Colony), Crete, France, Yugoslavia and possibly other European countries. It is the purpose of this paper briefly to survey the knowledge concerning this not too well known disease.

Definition

Tick paralysis is a disease of certain animals (colts, calves, dogs, cats, rabbits, sheep, pigs and grouse) and man consequent to the bite of ticks of various genera (Dermacentor, Ixodes, Haemaphysalis and possibly also Boophilus, Rhipicephalus, and Hyalomma) and is characterized by a most striking neurologic syndrome, consisting, in brief, of an ascending motor paralysis and incoordination with or without sensory changes. The disease can be produced experimentally at any time of the year, but in nature it is known to occur in the Northern Hemisphere between February and August.

Historical Aspects

Although the history of this disease in North America does not begin until the early part of the present century, its presence in Australia was recognized in the first quarter of the nineteenth century. Several observations on the effects of tick bite were recorded in the last century. A few of these specifically mentioned paralysis as the prominent feature of the disease in man and animals. With the beginning of the present century, investigators in South Africa reported a disease which produced motor paralysis in sheep. They attributed this disease to the bite of a certain tick. In North America attention was drawn to tick paralysis through the studies of Rocky Mountain spotted fever, then known to be caused by a tick bite. Tick paralysis was brought to light in Canada and the United States by Todd in 1912, who made a survey of the effect of tick bites in British Columbia and thus established the existence of tick paralysis as due to the bite of the tick Dermacentor andersoni Stiles, (then known as Dermacentor venustus). Following this, many experimental, entomologic and clinical studies were made which proved beyond a doubt the existence of this disease in both Canada and the United States. In more recent years similar studies in Europe have suggested its presence there and have thrown further light on its pathogenesis.

Etiologic Agents and Geographic Distribution

In North America two ticks, Dermacentor andersoni Stiles (wood tick) and Dermacentor variabilis Say (the common dog tick),

*Fellow in Neurosurgery, Mayo Foundation, Rochester, Minnesota.

are now known to cause tick paralysis. The wood tick flourishes throughout the Rocky Mountains as far south as New Mexico and Arizona and its northern boundaries are in southwestern Alberta and Saskatchewan and British Columbia. This tick is prevalent in California, Oregon, Washington, Idaho, Montana, Wyoming, southwestern North Dakota, the Black Hills of South Dakota and in Colorado, but so far tick parlysis has not been reported in California, Nevada, Arizona, New Mexico, Utah, North and South Dakota, nor from large portions of the other states in which the wood tick abounds. The reasons for this discrepancy have not become apparent as yet.

The common dog tick is prevalent in eastern and central United States and in California, but so far it has been indicated as the cause of tick paralysis in only a few instances in the Carolinas and Georgia.

The etiologic agents of tick paralysis in other countries are as follows: in Australia the tick Ixodes holocyclus Neumann; in South Africa the Ixodes pilosus Koch; in Crete the Ixodes ricinis Linn and possibly the Haemophysalis punctata. From France tick paralysis has been reported as caused by the native Ixodes hexagonus, while in Germany the disease has been experimentally produced by means of the tropical dog tick, Rhipicephalus sanguineas Latrielle. In Yugoslavia the so-called Shimteera disease in animals is probably identical with tick paralysis and is caused by several genera of ticks, Hyalomma, Rhipicephalus and Boophilus.

Symptomatology

The symptoms of this disease, though not rarely bizarre, usually run such a typical course that the mere thought of the disease together with the history should allow for an immediate diagnosis. The following description of the symptoms as they are likely to occur in a given case is taken from a recent review of the disease:[1]

"The victim frequently is irritable for twelve to twenty-four hours preceding the onset of paralysis. On awakening, the patient (usually a child) may or may not complain of pain or paresthesia and numbness in the lower extremities. He falls when he attempts to get out of bed but may be able to stagger to the breakfast table. The parents of the child soon find that he cannot feed himself because of incoordination (dysmetria and asynergia) and will feed him and put him to bed. Within the next few hours (frequently by 10 o'clock) flaccid paralysis of the lower extremities becomes evident with weakness of the upper extremities which becomes complete in another twelve to twenty-four hours if the tick is not removed. Bulbar paralysis is evidenced by dysphagia, dysarthria, lingual and facial paralysis, and ocular symptoms; finally death may ensue from respiratory paralysis.

"Examination of the patient reveals a flaccid type of paralysis with absent tendon and superficial reflexes; these reflexes have disappeared successively from below upward. The paralysis is usually bilaterally symmetrical as it ascends. Sensory changes, hypesthesia, anesthesia or dysasthesia may or may not be present singly or in combination. The pupils frequently are dilated and fail to respond to light or to accommodation, but they may be normal until the late stages of bulbar paralysis are manifest. When bulbar paralysis appears, the lower cranial nerves are found to be paralyzed first; thus the sternocleidomastoid, trapezius, tongue and pharyngeal muscles are affected first; the face and extraocular muscles soon follow, then respiratory paralysis and death occur.

"When the tick is removed before symptoms appear, either the paralysis or other symptoms will be mild and transient or they may not appear at all. If the tick is allowed to remain until bulbar signs are present, death from respiratory paralysis may ensue even though heroic supportive measures have been administered; this is particularly true of infants, for deaths have been reported among them after the removal of the tick even in an early phase of the disease."

Other symptoms are incoordination (apparently true ataxic movements), myoclonic twitchings and choreiform movements. The latter two are infrequently encountered.

Although the majority of the cases of atypical symptoms have occurred in Australia, a few have been encountered in this country. These can be enumerated as follows: (1) isolated nerve palsies in the region of the engorging tick, such as facial paralysis from a tick in the external auditory canal or behind the ear, paralysis of the frontalis and orbicularis oculi when a tick was present on the same

side of the forehead, paralysis of an upper extremity as a result of a tick attached over the brachial plexus; (2) other bizarre symptoms have become manifest irrespective of the location of the attached tick, such as blurring of vision, diplopia, nausea, dysphagia, various sensory changes and urinary retention.

Treatment

There is no more important aspect in the treatment of this disease than for the physician to think of tick paralysis whenever a patient presents himself with an ascending paralysis of Landry's type. Once this is accomplished, it is a simple matter to look for the tick concealed in the scalp (particularly of little girls with long hair), ears, axilla, under the breasts, in the groin, perineum and genitalia. Once the tick is located, its removal may be accomplished by one of several means such as gentle traction, application of heat, carbolated petrolatum, kerosene, gasoline, ether, or chloroform or by lifting off the tick with a hypodermic needle or dagger pointed scalpel. When the tick is removed, it is well to be certain that the mouth parts also have been completely removed. After removal of the tick, apply either silver nitrate or other antiseptic solution.

Except for the removal of the tick, treatment is entirely symptomatic. The usual dermatologic lotions are of value in those cases in which urticarial reactions develop from tick bite, but no known antisera have been shown to be of value except that produced in Australia for use in animals. The use of this serum for human beings is to be seriously questioned since severe urticarial and anaphylactic reactions have occurred after its administration to man.

Pathogenesis

Since the life cycle of the tick through the various stages of egg, larva and nymph are now well known and are available in both popular and scientific journals, it will not be reviewed. It is important to emphasize that the female tick only causes tick paralysis. The tick is brushed off from grasses, bushes and various low-lying shrubs onto the host and attaches itself to the host by means of its head parts or hypostome. It then begins the process known as engorgement—it feeds on the blood and lymph of the host, it increases in size and at the same time develops inside its own body several thousand eggs. Its weight increases from 1 mg. to 20 or 30 mg. by the fourth day of engorgement and up to 450 mg. or more by the sixth to tenth days, at which time engorgement becomes complete. The tick then falls off and deposits its thousands of eggs. Through some little understood mechanism, the tick develops a toxin in itself which it injects into the host usually between the fourth to sixth and subsequent days of engorgement.

The exact origin within the body of the tick of this toxin, called ixovotoxin by the Yugoslavian investigators, is not known, but certain investigations indicate that its origin may be from the ova or from the saliva or from both. Just why all anatomically identical ticks do not produce the toxin remains obscure nor are the extrinsic or intrinsic factors which may play a definite role in the formation of the toxin known. However, it has been shown conclusively that the causative agent of the disease is not of bacterial or virus origin.

Little is known concerning the manner in which the venom or ixovotoxin acts on the nervous system. However, from clinical and experimental data, it has been shown that this toxic substance acts in a selective manner on the anterior horn cells of the spinal cord, though occasionally the peripheral nerve and medullary nuclei also are involved. A few studies have been made at necropsy but as yet these have yielded only meager knowledge of the mysterious pathogenesis of this rapidly fatal disease.

Reference

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Remarks on Mechanisms in Non-Analytic Psychotherapy*

NORMAN REIDER, M.D.**
Los Angeles, California

I propose to discuss briefly a few of the mechanisms by which individuals overcome difficulties by means of psychotherapy other than psychoanalysis. The subject is so broad that I cannot possibly do more than scratch the surface of the therapeutic effects of external events, Christian Science, placebo medication, hypnosis, chiropractic and osteopathic treatments, and the techniques of the various psychological schools. The clinical and incontrovertible facts are that these methods and many others, often very unscientific, do help people. The question now is: How can so many unscientific methods produce therapeutic results?

The problem has both theoretical and practical importance, but is of special import to the physician who, accustomed in his own practice to attribute scientific validity to the pragmatic test of his therapeutic devices, finds himself faced by the dilemma of having to give credence to unscientific methods because they, too, have produced results. Often the tendency for the physician is to ignore or minimize the therapeutic effects of chiropractic treatment for an anxiety neurosis as something akin to "black magic" or quackery, and to let it go at that. Accustomed as he is to more rigid, scientific thinking, he cannot always comprehend the non-rational, magical, immature thinking of children and sick people.

Here, I believe, is the starting point of our understanding of the problem. Knight,[1] in his paper on "Why People Go to Cultists," points out that the following sequence of events takes place in individuals who are particularly addicted to magical thinking. All children, because of their lack of knowledge and comprehension of the real world about them, indulge in primitive thinking which is fantastic, unscientific, and superstitious. As the child matures he relinquishes this irrational type of thinking for more scientific and rational thinking influenced by education and realities. Hardly anyone, however, entirely overcomes all his magical thinking. Each of us to some extent has private superstitions or rituals upon whose magical power we occasionally rely.

It is important for us to recognize that when a person is ill or neurotic he tends to become more childish and relies more on his supposedly forsaken magical beliefs and wishes. In the process of maturing, which each child goes through, there is a period in which he believes his parents are omnipotent. He seeks and obtains from them love, security, and a feeling of well being. Parents, remembering their own childhood, intuitively or consciously, increase the child's belief in their magical omnipotence by telling them that they will kiss his wound and "it won't hurt any more" or by relieving the tension involved in pain and making the child feel better by caresses and embraces. And so an adult when he becomes ill experiences a revival of these childish beliefs in magic or childish fears in the face of which his adult habits of thought may be abandoned. He is ripe for the promise of cure of a patent medicine or a cultist who speaks reassuringly and authoritatively. Since a considerable proportion of the population remains at a childish level of thinking, to say nothing of its factual ignorance, it is not difficult to see that there is still an enormous response to magical medicines and magical therapeutics of all kinds.

The childish experience of feeling better after parental caressing and rubbing, and the traditional belief in "the laying of the hands" is exploited by those healers who promise relief through unscientific touching and rubbing measures. But what the cultist does unscientifically, physicians often fail to do with their mechanistic science. The cultist understands that he can satisfy these primitive childish needs and therefore is superficially correct in satisfying the desire for an omnipotent, magical assurance of cure. Simply put, the cultist meets the unconscious psychological need of the patient. But often it turns out that it is just a case of the blind

*Read at a Meeting of the Los Angeles Society of Neurology and Psychiatry.
**Now in the Armed Services of the United States.

leading the blind, of magic being used to combat magic. Parenthetically, I may say that I do not advocate physicians taking over the cultists' methods in treating these problems; there are more scientific approaches.

To be sure it is not the cultist alone who employs such magical methods. Physicians themselves often do so, quite wittingly at times and at other times actually believing, for instance, that there is something specific against the neurosis in the prescription they write. These methods do work in individuals in whom magical thinking still plays an important role. This is an abstract of Knight's thesis.

But even though dynamic psychiatry is a relatively young science, many physicians and individuals, whether they understand or not, influenced by popularizers, accept the thesis that at the basis of emotional disturbances lies a conflict. The concept of conflict is universally accepted by all schools of thought—by the religionist as a conflict between the forces of good and evil; by the organicist as a conflict between pathological processes and inherent homeostatic trends; by the Adlerian as a conflict between feelings of inferiority and compensatory mechanisms; by the Freudians as a conflict between biologically determined instinctual drives and opposing external forces or their internal representations. To repeat, the concept of conflict is universal and the more articulate patients are well aware of it and their awareness is embodied in their presenting complaint. I do not propose now to go into any theoretical discussion of the relative merits of the various schools of thought on this point, but I think we cannot avoid the conclusion that incompletely solved childhood conflicts breaking through form one basis for emotional disturbances. In other words, a person comes to a physician for help only when his own solutions of his conflicts no longer are adequate.

With the recognition of the psychogenic origin of emotional illnesses, the French school of psychiatry in the latter part of the nineteenth century began the psychogenic treatment of such illnesses. Then there developed the tendency to use suggestion and hypnosis as forms of treatment.

Recent studies on suggestion and hypnosis, especially by White[2] of the Psychological Clinic of Harvard University, tend to show that it is not true that the operating mechanism in suggestion and hypnosis is that they can cure those psychogenic illnesses which are auto-suggestive; the actual mechanisms are far more complicated than this simple formula would indicate. There is for one thing in the matter of suggestion, the going through of a ritual by the patient which connotes the same magical influence I have previously discussed. This is especially true where physicians prescribe daily routines which must be rigidly adhered to by the patient and they actually set up artificial compulsion neuroses, the compulsive element of which helps the patient to suppress or repress more successfully one element in the conflict disturbing him. This mechanism frequently can be seen and works out practically in many compulsive characters who begin having difficulties and go to physicians just for the strengthening of that particular type of defense which has failed them, namely, the compulsive ones.

Glover[3] and White point out that in the more formal suggestive methods, like hypnosis, the ritual is gone through by the physician with the same magical effect. The patient's role is the passive one and White proves that successful hypnosis can only occur in those individuals who have an infantile need for love, a tendency for passive compliance, and the wish to participate in omnipotence.

Have we not all had the experience of not giving any specific advice or suggestion and often, to our surprise and at times pleasure, the patient insists that he has been helped? So much emphasis has been placed upon the therapeutic effect of mental catharsis that some relatively simple mechanisms operating in such situations are overlooked. It is quite likely that the so-called cathartic effect which pictures the productions of the patient as poisons which he gets rid of by talking, produces its therapeutic effect because the therapist in such instances meets an unconscious need in the patient by listening patiently, not disapproving, and by persisting in a kindly attitude. Occasionally a patient will remark after the first or second interview that he wished he could have come to a parent with his problems earlier in life and have had them be non-critical and understanding. In such instances the therapist meets the need of the

patient to have a kind passive parent.

Following the reintroduction of hypnosis by the French schools, there occurred the development of the techniques of hypno-analysis, then psycho-analysis and the various divergent "analytic" schools. I should like to touch upon the therapeutic effects of the various psychological schools which employ exploratory and interpretive methods. Whether the type of treatment be called Adlerian, Jungian, Rankian, Stekelian, or distributive analysis or synthesis we are again faced with the peculiar situation that all of these schools, so far as theory is concerned, cannot be scientifically correct in toto, for many of their theories, while they have elements in common, do not agree in important details. How is it, then, that their practice often has therapeutic value? One answer is obvious: that the theory and the practice have nothing to do with each other; that their practice follows along intuitive lines of meeting the unconscious needs of patient in the manner mentioned above. Yet this cannot be entirely so. From case material it is brought out that often it is the interpretation to the patient of the reasons for his behavior which produces the therapeutic results. There is no doubt that at times the particular interpretation is correct and helps reveal to the patient motivating factors in his neurosis which had been outside of his conscious control.

Since it is not the purpose of this paper to discuss the therapeutic effect of correct interpretation I return to the problem of why incorrect or inexact interpretation has any therapeutic effect at all. Glover has treated this problem admirably in a paper written in 1931 in which he points out that suggestion by the mechanisms to which I have referred is operative. There are, however, other mechanisms involved in the very nature of the investigatory trend of the therapy. In many instances bringing out early data in the individual's life serves the purpose of stirring up guilt and when the therapist begins to indicate the nature of the origin of this guilt the repressive mechanisms are strengthened and the guilt and anxiety disappear.

I will illustrate briefly what often happens in such instances by case material. I had under treatment a sixty-two year old retired minister who had been suffering for years from insomnia and fatigue. In the course of going more deeply into historical data it became apparent that the motivating factors in this man's entire life were his attempts to avoid any aspect of real sexuality which he felt was dangerous. So much energy did he spend in fighting biologically determined needs that it was no wonder he was fatigued. He went into the ministry at an early age, studied hard, filled his mind repeatedly with "good thoughts" and did not marry until he was fifty years old. His insomnia was in a sense necessary for him to ward off disturbing dreams which occurred when his conscious controls diminished in sleep. When the inescapable meaning of his symptoms began to intrude upon his consciousness he became overwhelmed with anxiety and guilt, but had sufficient strength of character to marshal his defenses to a greater capacity than he had ever before done and successfully repressed what had really been bothering him. He left treatment free of symptoms, sleeping well and full of physical strength, denying that what had become apparent to him was in the slightest degree true. This therapeutic result was probably the best that could have been obtained in such an individual.

In other such individuals, especially the compulsive characters, whose need to intellectualize is great, the therapist actually helps the individual overcome difficulties by helping him intellectualize concerning himself. This again serves the purpose of aiding the repressive forces and submerging the disturbing instinctual drives or their derivatives. In these people a correct verbal formulation of their problem, something which these patients have not been able to reach themselves, is of great help. They feel that now they have something real to grapple with instead of something intangible.

I have noted that the investigatory trend in psychotherapy produces therapeutic results in still another way by an inexact application of psychoanalytic principles. I have reference particularly to problems in which feelings of guilt are very strong. In such instances incorrect interpretations concerning the guilt may have good therapeutic results when the therapist helps the patient to project his guilt onto his infantile life. Again, perhaps I can best illustrate what I mean by way of case material. A woman came for treatment because of severe anxiety and guilt feelings over

her marital situation. Her husband was kind to her, tolerant to a fault, yet she was not happy. She was irritable, plaintive, and felt exceedingly guilty because her dissatisfaction was so unjustified. In the course of investigation it came out that the patient's parents had been constantly quarreling with each other and that she was a spectator to many of their arguments. She was perplexed as to how two people who continued to live together could be constantly at dagger's point. In these arguments she sided with her mother and shared the hostile feelings towards her father. When these facts, many of which had been forgotten by the patient until brought out in the course of treatment, became clear, the interpretation, incomplete and inexact, was made to the effect that her present behavior was simply a reliving of her childhood situation and that she had learned of no other way to get along with a man except to fight with him and that therefore she need not feel guilty since her present guilt was unjustified, but belonged to a long-past part of her life. I say that this interpretation was incomplete and inexact because there was other material pointing very definitely to the fact that there was real cause for her to feel guilty in the present situation. Nevertheless, the interpretation had the effect of relieving her of her guilt by projecting it upon incorrect impression she had obtained from her parents. She was in a sense absolved; she was no longer at fault, but unsolved conflict in childhood was to blame. This type of "artificial paranoia" may frequently have such a partial solution.

Occasionally we see poor results in the treatment of psychopathic personalities to whom the same type of inexact interpretation is given whereby they no longer have anxiety over their condition but remain unchanged if not actually worse because they can point an accusing finger at their unhappy childhood as a cause of their condition.

Summary

It appears that unscientific methods of approach to emotional disturbances can and do help patients get over their difficulties. They accomplish their therapeutic results by: (1) meeting the unconscious needs of the patients. These needs are infantile yearning for love, a desire to participate in omnipotence, and at times (though I have given no example of this in my paper), the wish for punishment. (2) The therapist, by suggestion or inexact interpretation, often succeeds in repressing one of the elements which constitutes the conflict. In doing so, artificial neuroses are often set up but are much more acceptable to the patient because the scene of the conflict is shifted or displaced. In the investigatory types of psychotherapy, guilt is often alleviated by helping the patient displace it onto childhood events so that the patient no longer feels the guilt as belonging to his present situation.

There are undoubtedly other mechanisms operative which are probably as important as those I have discussed. There are the mechanisms of recovery where no treatment is given, which in itself is a very interesting field of study. I hope that this brief exposition may be an impetus towards the adoption of a scientific attitude towards this important subject. Practically, there will be an important end achieved if the physician becomes aware that these unscientific methods lead to cures and that he could obtain the same if not better results by more scientific methods.

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CASE 84255

Presentation of Case

A 55-year-old, unmarried, white man was admitted to the Psychiatric Service of the Cincinnati General Hospital on December 29. At no time was a satisfactory history obtained. The only point upon which the patient's brother and sister agreed was that he had drunk alcoholic beverages heavily for an unknown period. Two weeks before admission the patient fell down two flights of stairs. The only damage sustained appeared to have been a bruised hip. He had complained of headache frequently after the fall, and had continued to drink excessively. When sober he was thought to have acted peculiarly. No definite delusions or hallucinations were noted by the relatives.

Examination—On admission he was markedly emaciated, dehydrated, and evidently chronically ill. He mumbled to himself and usually became abusive when questioned. At times he complained of low back pain and pain in the shoulders, particularly the right, and also in the arms. He was not oriented, and he seemed surprised when he heard he was in the hospital. His speech was incoherent as a rule, although sometimes, particularly when struggling with his restraints, he would utter such phrases as, "I'll get it off some way," "I'm trying to remove that cuff," "I put this shackle on too tight." He struggled against his wrist shackles frequently, although occasionally he could be trusted without them. He expectorated in bed and on the floor, and urinated in bed. He would reach in the air for what must have been illusory objects.

The abnormalities on admission examination besides the psychotic state were inequality of the pupils, the left measuring 6 mm. in diameter, the right 4; the right reacting through a fair range, and the left through a poor range to flashlight. The abdominal reflexes and the tendon reflexes in the lower extremities were not obtainable. The neck was somewhat stiff. Many hard lymph nodes were palpated in both inguinal regions. There was a palpable node in the right axilla. Blood pressure was 130/110.

Course—The degree of delirium altered unaccountably during the hospital course of 16 days. For short periods he appeared to be relatively clear and during these times he would feed himself, eat well, and would respond pleasantly. From the second to the fifth hospital day, he complained of severe epigastric pain, and on the tenth hospital day he said in a relatively clear moment, "I've hardly ever been sick until now."

For the most of the hospital course he was irrational and uncooperative. At times he would refuse nourishment or would exhibit belligerency, talking and screaming as though he were conversing with an hallucination. He would become quite excited, and on one occasion he bit an orderly. At times he was drowsy and difficult to arouse. On several occasions he was seen to masturbate.

On his thirteenth hospital day the following neurological abnormalities were noted: The left pupil was 6.5 mm. in diameter, the right 4. Again the right reacted fairly well to light, the left very poorly. The disc outlines were slightly blurred, particularly the nasal margins. The neck resisted any passive movement and there were positive Kernig signs, bilaterally. There was no localizable weakness There was generalized loss of muscle. There was tenderness to deep pressure in the muscles of the legs and feet. The abdominal reflexes and the knee jerks and ankle jerks were not obtained. There was a left Chaddock; otherwise, the plantar responses were flexor in type.

The temperature which had been ranging around 99° F. rose abruptly on the fourteenth hospital day. At this time signs of lobar pneumonia were found. The temperature continued to ascend along with the pulse and respirations until shortly before death when

the temperature was recorded at 103° F. rectally, and the pulse 160, and the respirations 50. The pulse had been rapid throughout the hospital course, ranging between 100 and 120 beats per minute.

On the thirteenth hospital day, he had what appeared to be the first of a series of seizures. These were tonic in nature, the patient stiffening, and staring straight ahead and breathing heavily. He seemed unable to speak during these episodes which lasted for about 5 minutes. These spells were observed infrequently during the remainder of his life. He continued to groan, claw at the air, and talk irrationally. These active periods would be interrupted by episodes of quiet drowsiness. He died with signs of pneumonia on January 13, after sixteen days in the hospital.

Laboratory Data—The analysis of the blood was within normal limits until the onset of pneumonia. The hemoglobin was estimated to be between 85 and 95 per cent, the blood count was 4.5 million, the white blood count was 9,000. Three blood Kahns were negative. The patient was incontinent. A urinalysis performed on January 1 was completely normal, the specific gravity of the urine was 1026. On December 30, a roentgenogram of the chest was reported as follows: "Bony framework, heart, midline structures, and diaphragm are normal. The lung fields are clear."

Seven analyses of the cerebrospinal fluid were done, and are abstracted. The figure 250+ indicates that the fluid ran over the top of a single manometer.

Differential Diagnosis

Dr. Joseph P. Evans: It seems that the patient was strikingly emaciated, dehydrated, and chronically ill. The appearance of chronic illness and emaciation are significant points. The inequality of the pupils, the left 6 mm. in diameter, the right 4, the right reacting through a fair range, the left through a poor range, brings up the question of trauma, and from the localizing point of view would suggest involvement of the third nerve. The abdominal and tendon reflexes were not obtainable, the neck was stiff. The significance of the absent abdominal reflexes at this age is difficult to determine. The lack of tendon reflexes may be related to the alcoholism, and suggests a degree of peripheral neuritis associated with it. Inability to obtain ankle jerks in bed in older people is not particularly significant. Stiffness of the neck would suggest a meningeal disorder. The palpable lymph nodes may well be a diagnostic point; one would like a description of their characteristics.

During the hospital course the delirium altered unaccountably for 16 days, there was variance in the level of awareness. There was on one occasion severe epigastric pain, and in a clear moment he stated that he had hardly ever been ill. This is difficult to interpret, but some significance may be attached to the epigastric pain. The examination and course indicate an organic psychosis. Two weeks after admission there was the same inequality of the pupils and some blurring of the nasal margins of the optic discs. The neck was stiff, which would indicate meningeal reaction. There was general loss of muscle, which is interesting because again it suggests chronic disease. Tenderness of the legs and feet, and the loss of deep reflexes suggest peripheral neuritis.

The seizures are of considerable interest from the physiological standpoint. The at-

Date	*Pressure*	*Color*	*Lymphs.*	*Mono.*	*Polys.*	*RBC*	*Protein*
12/29	250+	water	15	35	0	0	195
12/31	70	cloudy yellow	19	32	10	0	
1/4	"normal"	sl. xantho.	21	80	4	0	270
1/10	250	sl. xantho.	20	40	0	0	250
1/11	250+	sl. xantho.	20	20	0	1330	200
1/12	250+	bloody xantho.	75	0	75	4050	220
1/13	320	very xantho.	75	65	10	1350	140

The sugar and chloride contents of these fluids were normal; the gold curves, Wassermanns, and cultures were negative. The House Officer who counted the cells in the final cerebrospinal fluid noted that the great majority, if not all, were very large, and that these large cells contained a reticulated nucleus.

tacks were tonic in nature, there being a number of these episodes which may have represented some form of decerebrate attack. These tonic attacks suggest posterior fossa or midbrain localization.

Aside from the clinical examination, I would think the CSF of great importance. Obviously one is dealing with a disease which causes increased intracranial pressure. It is interesting that the fluid became progressively xanthochromic throughout the hospital stay of two weeks, and that is suggestive either of a superficial cortical process or meningeal reaction accompanied by an outpouring of the protein into the CSF; or of hemorrhage. I would suspect that the increased protein content with blood appearing late may well mean that there was a pathological process going on within the meninges. One has to interpret the cell content, a point which I think is of great significance in this case. The House Officer, who counted the cells, found that they were quite large and that they were reticulated.

I feel then that this man was a chronic alcoholic, and that he had obvious trauma, though perhaps not involving the head. However, minor injuries may be productive of subdural clot. The possibility of a neoplastic process which had antedated his obvious difficulties must be suspected in an older individual entering the hospital in an emaciated condition. I am, at this point, undecided between the diagnosis of subdural hematoma, or some form of neoplasm; of the two the latter would be a bit easier to explain. The cell picture in the CSF argues very definitely against subdural hemorrhage and I think that diagnosis unlikely. If this be a localized neoplastic process, I would suppose that its localization might well be in the frontal region on either side; the left frontal region rather than the right, taking the oculomotor signs into consideration. But if localized, it must be putting cells into the CSF.

One should comment on the fact that there was pain in the back and shoulders which may accompany a meningeal process. The glandular enlargement might indicate some form of leukemia. The blood content, however, is normal. The epigastric pain is hard to interpret.

I am inclined to think that this man had a neoplastic process which was causing diffuse leptomeningeal reaction and which gave evidence of its presence in the cellular content of the CSF, and which blocked the CSF pathways. An abscess is unlikely. It is my belief that diffuse neoplastic involvement accounted for the psychotic state and gave rise to pupillary changes and increased intracranial pressure. The absence of other cranial nerve signs is important, but shouldn't be allowed to discount the diagnosis. The evidence of emaciation is the significant clue.

Dr. Milton Rosenbaum: Doctor Evans mentioned localization in the frontal region. I should like to ask him what sort of neoplasm he thinks this is and why he thinks he can localize it.

Dr. Evans: There is very little localizing evidence, mostly pupillary signs. I would think that the disease is more likely to be diffuse sarcomatosis.

Dr. John Romano: In respect to the explanation for the absence of reflexes, if the patient had a diffuse, meningitic, neoplastic process, involvement of the cord and roots might account for this sign.

Dr. Rosenbaum: From the differential diagnostic point of view, it is common to have pupillary changes in the delirium associated with alcoholism. However, the pupillary changes from day to day are not consistent. I think, therefore, it is extremely significant that the pupils didn't change, and in view of what has been said, evidently he had a peripheral lesion of the third nerve, possibly due to carcinomatosis of the base of the brain. The pupils as described are not the usual alcoholic pupil.

Student: I would like to know about emaciation in association with alcoholism.

Dr. Charles D. Aring: Does anyone think that persons addicted to alcohol are usually emaciated?

Dr. Rosenbaum: No, I don't think so.

Student: If this is neoplasm, was the onset of symptoms initiated by the trauma?

Dr. Aring: Whether or not trauma lights up infection or neoplasm of the central nervous system is a point in argument. I think your opinion is as good as ours.

Clinical Diagnosis

Subacute leptomeningitis, undetermined origin.

Dr. Evans' Diagnosis

Diffuse sarcomatosis of leptomeninges.

Anatomical Diagnosis

Carcinomatosis of leptomeninges; adenocarcinoma of the kidneys.

Pathological Discussion

Dr. I. Mark Scheinker: On gross examination of the brain, there were no abnormalities to be seen except for slight opalescence of the leptomeninges, and small areas of subarachnoid hemorrhage. Microscopically the leptomeninges were diffusely infiltrated by tumor cells. The cells were epitheloid in nature and appeared to arrange themselves in alveolar-like structures. They revealed a marked polymorphy. Numerous mitoses could be seen.

The entire leptomeninges were infiltrated by these neoplastic cells. It is to be emphasized that the leptomeninges were not thickened, which is an important point because the process is apt to be overlooked during the gross examination of the central nervous system. A second important point is the absence of any secondary inflammatory reaction (no lymphocytes or leukocytes), and the presence of slight subarachnoid hemorrhage. It is interesting that the parenchyma of the central nervous system did not show much change. Rarely small vessels in the cortical tissue subjacent to the pia were surrounded by cuffs of neoplastic cells.

The spinal cord tissue was well preserved, but the leptomeninges were infiltrated by the neoplastic cells which could be seen around the anterior and posterior spinal roots. The strangulation of roots was undoubtedly responsible for some of the spinal root symptoms (pain, areflexia). The absence of inflammatory reaction again should be noted; there were no metastases within the spinal cord tissue proper.

On the base of the brain stem one found the third nerve infiltrated and partially destroyed by the same cells seen in the leptomeninges.

The kidneys showed infiltration with the same type epitheloid cell, which was interpreted as kidney carcinoma. No tumor was discovered elsewhere, which was surprising, particularly since primary kidney carcinoma is rare.

The final diagnosis was diffuse, metastatic carcinomatosis of the leptomeninges. This diffuse carcinomatosis usually is not associated with the formation of solid tumor metastasis. In spite of the fact that the leptomeninges were involved uniformly, there was no secondary inflammatory reaction in the leptomeninges.

There are a few correlary points to be made between the pathological and the clinical findings. Generally the symptoms of leptomeningeal carcinomatosis are characterized by irritative meningeal phenomena, and by cranial nerve palsies and areflexia due to strangulation of cranial nerves and spinal roots.

If we bear these symptoms in mind and pay some attention to the differentiation of the cells that sometimes are found in the CSF, the diagnosis can be made before death. Tumor cells in the CSF are too often thought to be white blood cells. There is usually an absence of cerebral focal lesions such as massive hemiplegia or aphasia in leptomeningeal carcinomatosis.

Dr. Aring: I think it is worth re-emphasizing the value of the CSF examination, and the necessity of careful analysis of cells therein. If the CSF examination is well and accurately done, one can lean on it heavily. As a single maneuver, it ranks next in importance to history in the neurological work-up. One should never trust the word "normal" as a measure of CSF pressure; the operator either records the exact figure or obviously he knows nothing about the pressure. Cell counts, such as those listed in the protocal, should be viewed with a high degree of suspicion. For example, a count of 50 per cent polymorphonuclear leukocytes in 150 cells, late in any central nervous disease (see CSF examination of 1/12), is practically unknown. I would urge that the CSF be centrifuged and stained with Wright's stain, when there is doubt as to the identification of the cellular content with the Unna polychrome methylene blue technique, in the blood counting chamber.

Dr. Romano: About the carcinomatosis, I think one important point clinically is the sudden onset which may be confused with a vascular phenomenon. A second point is that one has seen patients with carcinomatosis

who had involvement of the leptomeninges and hemiplegia. On section, one patient had a large discrete mass in the brain. In two patients that I recall the CSF study showed no tumor cells, and they had tremendous clinical and histological evidence of leptomeningeal involvement throughout the neuraxis. About the CSF examination, if it is inaccurately performed, it may throw you off and would be better left undone. One should draw the attention of someone else to the cells that cannot be interpreted.

Dr. Aring: Would anyone like to render a hypothesis as to why metastases involve the leptomeninges or the brain, one to the practical exclusion of the other?

Dr. Scheinker: One hypothesis is that if the invasion of the nervous system occurs via the lymph stream, the leptomeninges (subarachnoid space) and not the cerebrum is involved. On the other hand if the carcinoma spreads via the blood stream, the primary invasion involves the cerebrum in the form of solid tissue. Personally, I cannot follow this argument.

Dr. Rosenbaum: Was there a mass in the kidney? The urine was normal.

Dr. Scheinker: The kidneys appeared grossly normal, and together weighed 360 grams. Coronal sections revealed numerous yellow-white, firm, bulging areas rather square in shape and measuring approximately 2x2 cm. They varied in size and all of them were confined to the cortical portion of both kidneys.

I think the primary site was arrived at by exclusion. No other primary source could be found and the only extraneural tumor that was found involved the kidneys, so it was concluded they must be the primary site of the tumor formation. A small primary tumor which may give rise to tremendous damage elsewhere (metastases) may be overlooked. Evidently the glands were not considered of sufficient size to require histological examination, particularly since the pathologist had no inkling of the nature of the disorder before microscopy.

Book Reviews

AUTONOMIC REGULATIONS. Their Significance for Physiology, Psychology and Neuropsychiatry, by Ernst Gellhorn. Interscience, New York, 1943. Pp. 373 with 80 illus. $5.50.

There is an enormous amount of information in this compact volume. Gellhorn has listed more than 1100 separate references. But it is not a mere compilation, a presentation of conflicting statements, such as characterizes so much of the present literature on the autonomic system. Much more it represents a brilliant study of the conflicting results in terms of measurable differences, harmonized on the basis of exact experiments that reveal clearly the causes for such inconsistencies. Furthermore, the work indicates the extremely complicated mechanisms that go into the general picture of homeostasis, the interactions of the adrenal-sympathetic and vagus-insulin systems at the various levels from the cortical through the hypothalamic to the bulbar, spinal, peripheral and intermediate mechanisms. Gellhorn is at pains to show that the systems react not only within their own sphere, but also among themselves, and upon the organism as well as upon the organ. In the course of this demonstration he cites many experiments carried out in his own laboratories, thereby revealing first hand knowledge of his subject matter.

For the neuropsychiatrist the volume is rather heavy reading. The author follows the physiologist's tradition in sparing words and while the argument is clear, still the various results and conclusions follow each other in such rapid succession that the leisurely-minded reader is apt to lag behind the author's meaning. In other words, the book is to be studied rather than read if the maximum benefit is to be obtained.

As far as psychiatry is concerned, Gellhorn finds that the importance of the shock therapies lies in the common ground of perturbations of the autonomic centers provoked by insulin, metrazol, the electric current, narcotics, fever, thyroxin—all of which seem to differ in many respects from one another. The author admits, naturally, that he is "fully aware of the fact that the problems of schizophrenia has many more facets than can be discussed in a book on 'Autonomic Regulations'," and he pays somewhat more than lip service to the idea that cortical thought processes can influence the reactions of the autonomic system. He does well, however, merely to mention these things and to keep his feet on the ground and his eye on the kymograph. On the whole the volume represents a masterly exposition of the complexities of the autonomic system and of their interactions of the various components in the preservation of an alert and well integrated organism.

Walter Freeman.

BORDERLANDS OF PSYCHIATRY. Cobb, S. Harvard University Monograph in medicine and public health No. 4. Harvard University Press. Cambridge: 1943. Pp. 166. $2.50.

This is a most delightful and satisfying book to read for anyone interested in medicine, psychiatry, sociology or psychology. Well written, pleasingly informative and adequately instructive, it deals with those human problems always regarded as primarily medical but consistently neglected because they are of a borderland character and have never been adequately surveyed from the medical point of view. Speech defects, psychoneuroses, epilepsy, minor neurological handicaps, the relationship between psychological states and body manifestations, all these and more are treated simply, informatively and so clearly that even the reader of slight training is certain to derive benefit. As for the general practitioner of medicine, this book certainly clarifies innumerable problems and answers many questions frequently and disappointingly encountered. The book deservedly belongs on every medical desk for ready consultation and should be required reference reading for every college student interested in human problems. More than that, it is delightful reading and will prove invaluable in answering those questions so frequently asked of the general practitioner.—M. H. Erickson (Eloise Hospital).

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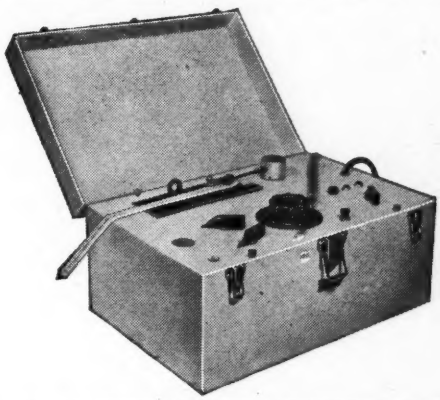

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